IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Applicant: Jay A. Goldstein, Michael Rothman, and Whe-Yong Lo

Serial No.: 10/691,928 Art Unit: 1616

Filed: October 23, 2003 Examiner: David Paul Stitzel

For: *ANTIFUNGAL FORMULATIONS*

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

DECLARATION UNDER 37 C.F.R. § 1.132

Sir:

I, Jay A. Goldstein, hereby declare that:

1. I am a co-inventor of the above-identified application. I have been a licensed physician since July 1973. I began my medical career as an Emergency Physician, and practiced this specialty for four years. In September of 1977, I started training in Dermatology, and became a fully trained Board Certified Dermatologist in November of 1980. My CV is attached as Exhibit A.

2. During my long medical career, both as an Emergency Physician, and as a Dermatologist, I have found that rashes, and particularly inflammatory tinea (ringworm) were a particularly common and often stubborn problem to treat. Such rashes respond to topical anti-fungals, but in a very slow fashion. It can take up to 4-6 weeks for these rashes to clear and for the patient to be symptom free. Even as the rash fades, the patient is still often bothered by

intense, unremitting itching, burning and discomfort. There was, and is a product, Lotrisone, which was developed to both treat the tinea, as well as the accompanying inflammation and itching, which were often the main reasons that the patient sought medical attention. Lotrisone was a combination of anti-fungal clotrimazole with a high potency corticosteroid, betamethasone dipropionate. This drug was effective in clearing the tinea, as well as rapidly decreasing the itching, which without the steroid, would normally last up to several weeks. With Lotrisone, however, the itching component would often disappear within days, making the patient more comfortable. The problem with Lotrisone, however, was that the steroid was too potent to be used safely on thin skinned area of the body, and thus often caused stretch marks, thinning of the skin, as well as other changes.

3. Because of my many years of experience both as an Emergency Room Physician, and as a Dermatologist, I saw the need for a preparation which would address both the fungal infection, as well as the intense itching and inflammation associated with the fungal infection. While others thought that perhaps slightly lower potency or even higher potency steroids would be acceptable, I felt that any steroid other than those safe for use on the face and other thin skinned area would not be appropriate. Of course, there was the risk that lower potency steroids would not be effective. I began using anti-fungal preparations in conjunction with low potency topical steroids on my patients with inflammatory tinea, and found that in fact such preparations were both safe and effective. They shortened the time to clearing of the fungus, and they dramatically decreased the symptoms of redness and especially itching. It would have been

unethical to compare the type of products that I used with compounds using stronger, more potent steroids, as there would be the real risk of major untoward side effects.

4. I have developed a formulation that that rapidly clears both their fungus, and their associated symptoms of itching and inflammation. This is further demonstrated by studies conducted using topical compositions containing a combination of an antifungal agent in combination with a low to mid potency anti-inflammatory steroid in the treatment of fungal diseases and their related inflammation, especially for conditions such as tinea cruris, intertriginous dermatitis, and tinea corporis.

5. Case Report

Patient: C.S, 74 y.o. White male

History of Present Illness: Long standing recurrent tinea cruris of inguinal folds.

Initial Treatment: None

Physical Examination: Erythema with scale in inguinal folds.

Diagnosis: Tinea Cruris

Treatment: Clotrimazole 1% cream with alclometasone dipropionate 0.05% cream applied twice daily.

Results: Complete clearing after several weeks of usage.

6. Case Report

Patient: B.T. 72 y.o. White female

History of Present Illness: Several days of pruritic inflamed eruption beneath right breast.

Prior Treatment: None

Physical Examination: Erythematous dermatitis beneath right breast.

Diagnosis: Intertriginous Dermatitis.

Treatment: Oxicanozole cream 1% with Hydrocortisone cream 2½% applied twice daily.

Results: Marked clearing at seven days.

7. Case Report

Patient: D.E. 52 y.o. White male.

History of Present Illness: Two months of very pruritic eruption beginning on left foot, spreading to right hand.

Initial Treatment: None

Physical Examination: Well-defined, annular, scaly, erythematous, inflamed eruption on dorsum surface left foot, with similar plaque on right hand.

Diagnosis: Tinea Corporis

Treatment: Econazole cream 1% with fluocinalone acetonide cream 0.01% applied twice daily.

Results: Marked decrease of pruritus within 3 days. Eruption essentially cleared at 3 weeks.

8. Case Report

Patient: M.B. 61 y.o. White male

History of Present Illness: Eruption of right and lower leg of several months duration. Known history of "tinea."

Prior Treatment: None

Physical Examination: Plaques of annular dermatitis of lower legs, right greater than left. 10 toenail onychomycosis.

Diagnosis: Tinea corporis, with tinea pedis and onychomycosis.

Treatment: Econazole cream 1% with alclometasone dipropionate 0.05%, applied twice daily.

Results: Marked clearing at 3 weeks, but with some residual eczematous changes still present.

9. Case Report

Patient: R.B. 62-year old white male.

History of Present Illness. Eruption began on right lower leg in mid-August. No response to topical steroids.

Physical Examination: Raised annular eruption on right lower leg.

Laboratory. Biopsy on September 26, 2005 revealed hypersensitivity reaction.

Additional Treatment. High potency steroids again prescribed without effect.

Additional laboratory Test. Special stains revealed inflammatory tinea.

Treatment: Application twice daily of desonide cream and clotrimazole cream together resulted in essentially complete clearing within two weeks.

10. In summary, oxicanozole cream 1% with hydrocortisone cream 2½% applied twice daily and econazole cream 1% with fluocinalone acetonide cream 0.01% applied twice daily resulted in marked clearing of pruritus and the eruption at 3 weeks. Clotrimazole 1% cream with acalmetasone dipropionate 0.05% cream applied twice daily was effective in completely clearing long standing recurrent tinea cruris, after several weeks of usage. Econazole cream 1% with alclometasone dipropionate 0.05% applied twice daily resulted in marked clearing of eruption in a patient with a history of tinea.

11. Enclosed with this declaration are colored photographs showing bright red erythematous eruptions on a patient's leg before treatment (labelled "Before Treatment") and the patients leg after treatment with desonide cream and clotrimazole cream (labelled "After Treatment") along with the patient record (Exhibit D). No response had been obtained with topical steroids. Upon initial examination of the patient, high potency steroids were again prescribed without effect. Application of a combination of desonide cream and clotrimazole cream (twice daily) resulted in marked improvement in five days and essentially complete clearing within two weeks.

12. The compositions used in the examples above have advantages over other compositions which contain very potent steroids such as betamethasone and dexamethasone (see Goodman and Gilman's The pharmacological Basis of therapeutics, 9th edition, 1996, p1466, attached as exhibit B) associated with severe side effects. It is undesirable to use mid-potency or higher potency steroids for topical treatment for extended periods of time because of associated risks. The compositions exemplified above employ low potency, Class 6 steroids (see attached (Exhibit C) potency chart of steroids listed by the National Psoriasis Foundation), i.e. fluocinalone acetonide, alclometasone dipropionate, desonide, and hydrocortisone 2 ½%. Other commercialized products have utilized only 1% hydrocortisone, which is too low in potency to have significant anti-inflammatory properties. We utilize prescription strength steroids that are safe for all parts of the skin, are safe for extended periods of use, but have superior potency as compared to OTC products.

13. I declare that all statements made herein of my own knowledge and belief are true and that all statements made on information and belief are believed to be true, and further, that the statements are made with the knowledge that willful false statements are punishable by fine or imprisonment, or both, under section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Date: 2/26/07

Jay A. Goldstein

EXHIBIT A

U.S.S.N. 10/691,928
Filed: October 23, 2003
DECLARATION UNDER 37 C.F.R. § 1.132

Curriculum Vitae

Identifying Information	Jay A. Goldstein, M.D. 31 Claremont Street Newton, Massachusetts 02158
Office Address	67 Union Street – Suite 501 Natick, Massachusetts 01760
Date and Place of Birth	January 9, 1947 Paterson, New Jersey
Citizenship	U.S.A.
Pre-medical Education	Boston University School of Medicine 1968 – 1972 M.D., 1972
Internship	Herrick Memorial Hospital Berkeley, California 1972 –1973 Rotating
Residency	Boston University Medical Center Dermatology 1977 – 1980
Licensure	Massachusetts #39484 Rhode Island #9865
Certification	American Board of Dermatology, 1980
Professional Societies	American Academy of Dermatology Society of Investigative Dermatology
Academic Appointments	Boston University School of Medicine, Associate in Dermatology
Hospital Appointments	Metrowest Medical Center Natick, Massachusetts Boston Medical Center Boston, Massachusetts
Publications	1. Goldstein JA and Pochi PE: Failure of Benzoyl Peroxide to Decrease Sebaceous Gland Secretion in Acne Dermatologica 162: 287-291, 1981

Curriculum Vitae

2. Shalita AR, Strauss JS: Comparative Effect of Isotretinoin and Etretinate on Acne and Sebaceous Gland Secretion. Journal of the American Academy of Dermatology 6: 760-765, 1982

3. Goldstein JA, Comite H, Mescon H, Pochi PE: Isotretinoin in the Treatment of Acne: Histologic Changes, Sebum Production, and Clinical Observations. Archives of Dermatology 118: 555-562, 1980

4. Goldstein JA, Pochi PE: Adolescent Acne. The Female Patient 5: 57-66, 1980

5. Barza M, Goldstein J, Kane A, Feingold DS, Pochi, P: Systemic Absorption of Clindamycin Hydrochloride After Topical Application. Journal Amer Acad Dermatol 7: 208-14, 1982

Exhibit B

GOODMAN & GILMAN'S The PHARMACOLOGICAL BASIS OF THERAPEUTICS

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Illustrations by Edna Kunkel

GOODMAN & GILMAN'S The PHARMACOLOGICAL BASIS OF THERAPEUTICS

Ninth Edition

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Goodman and Gilman's THE PHARMACOLOGICAL BASIS OF THERAPEUTICS, 9/e

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This book was set in Times Roman by York Graphic Services, Inc. The editors were Martin J. Wonsiewicz and Peter McCurdy; the production supervisors were Robert Laffler and Clare Stanley, and the cover designer was Marsha Cohen/Parallelogram. The index was prepared by Irving Condé Tullar.
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Corticosteroids are grouped according to their relative potencies in Na^+ retention, effects on carbohydrate metabolism (*i.e.*, hepatic deposition of glycogen and gluconeogenesis), and antiinflammatory effects. In general, potencies of steroids as judged by their ability to sustain life in the adrenalectomized animal closely parallel those determined for Na^+ retention. Potencies based on effects on glucose metabolism closely parallel those for antiinflammatory effects. The effects on Na^+ retention and the carbohydrate/antiinflammatory actions are not closely related. Based on these differential potencies, the corticosteroids traditionally are divided into mineralocorticoids and glucocorticoids. Estimates of potencies of representative steroids in these actions are listed in Table 59–2. It should be kept in mind, however, that a number of steroids that are predominantly classified as glucocorticoids, such as cortisol and prednisone, also possess modest but significant mineralocorticoid activity. Clinically significant changes in fluid and electrolyte handling can result from the mineralocorticoid effects of these "glucocorticoids." In contrast, aldosterone is exceedingly potent with respect to Na^+ retention but has only modest potency for effects on carbohydrate metabolism. At normal rates of secretion by the adrenal cortex or in doses that maximally affect electrolyte balance, aldosterone has no significant glucocorticoid activity and thus acts as a pure mineralocorticoid.

General Mechanisms for Corticosteroid Effects. Corticosteroids interact with specific receptor proteins in target tissues to regulate the expression of corticosteroid-responsive genes, thereby changing the levels and array of proteins synthesized by the various target tissues (see Figure 59–5). As a consequence of the time required for changes in gene expression and protein synthesis, most effects of corticosteroids are not immediate, but become apparent after several hours. This fact is of clinical significance, because a delay generally is seen before beneficial effects of corticosteroid therapy become manifest. Although corticosteroids predominantly act to increase expression of target genes, there are well-documented examples where glucocorticoids decrease transcription of target genes, as discussed below. In contrast to these genomic effects, recent studies have raised the possibility that some actions of corticosteroids are immediate and are mediated by membrane-bound receptors (Wehling, 1994).

Through the use of molecular biologic approaches, the receptors for the corticosteroid hormones have been cloned and their structures determined. These receptors are members of a superfamily of structurally related proteins, the nuclear receptors, that transduce the effects of a diverse array of small, hydrophobic ligands, including the steroid hormones, thyroid hormone, vitamin D, and retinoids (Mangelsdorf *et al.*, 1994). These receptors share two highly conserved domains: a region of approximately 70 amino acids forming two zinc-binding domains, termed *zinc-fingers*, that are essential for the interaction of the receptor with specific DNA sequences, and a region at the carboxy terminus that interacts with ligand (the ligand-binding domain). Removal of the ligand-binding domain from the glucocorticoid receptor leads to its constitutive activation (*i.e.*, activation in the absence of ligand), suggesting that the glucocorticoids activate their receptor by relieving the inhibitory influence of the carboxy-terminal region.

Table 59–2
Relative Potencies and Equivalent Doses of Representative Corticosteroids

COMPOUND	ANTI-INFLAMMATORY POTENCY	Na^+-RETAINING POTENCY	DURATION OF ACTION*	EQUIVALENT DOSE†, mg
Cortisol	1	1	S	20
Cortisone	0.8	0.8	S	25
Fludrocortisone	10	125	S	‡
Prednisone	4	0.8	I	5
Prednisolone	4	0.8	I	5
6α-methylprednisolone	5	0.5	I	4
Triamcinolone	5	0	I	4
Betamethasone	25	0	L	0.75
Dexamethasone	25	0	L	0.75

* S, short (*i.e.*, 8–12 hour biological half-life); I, intermediate (*i.e.*, 12–36 hour biological half-life); L, long (*i.e.*, 36–72 hour biological half-life).

† These dose relationships apply only to oral or intravenous administration, as glucocorticoid potencies may differ greatly following intramuscular or intraarticular administration.

‡ This agent is not used for glucocorticoid effects.

Topical steroids

Potencies of topical steroids

Topical steroid medications come in various strengths, ranging from very strong, or superpotent (Class 1), very weak, or least potent (Class 7). Once a person has stopped responding to a steroid of a particular strength or potency, it is unlikely he or she will respond to any brand of steroid at an equal or lower streng unless an extended period of time has elapsed. The potency chart below provides the potencies of a varie of steroid medications used to treat psoriasis.

Generally, the stronger the steroid, the more effective it is in clearing psoriasis, but the risk of side effects also greater. The base, or formulation, of a steroid medication can also influence how much medication penetrates the tissue. Steroids come in a variety of bases, such as creams, ointments, gels, sprays, solutions, lotions, foam and tape.

Potency chart

The following potency chart categorizes brand-name topical steroid medications along with the name of th corresponding generic drug. The list positions these medications according to their potency. The list may n be comprehensive.

BRAND NAME	GENERIC NAME
CLASS 1 - Superpotent	
Clobex Lotion, 0.05%	Clobetasol propionate
Cormax Cream/Solution, 0.05%	Clobetasol propionate
Diprolene Gel/Ointment, 0.05%	Betamethasone dipropionate
Olux Foam, 0.05%	Clobetasol propionate
Psorcon Ointment, 0.05%	Diflorasone diacetate
Temovate Cream/Ointment/Solution, 0.05%	Clobetasol propionate
Ultravate Cream/Ointment, 0.05%	Halobetasol propionate
CLASS 2 - Potent	
Cyclocort Ointment, 0.1%	Amcinonide
Diprolene Cream AF, 0.05%	Betamethasone dipropionate
Diprosone Ointment, 0.05%	Betamethasone dipropionate
Elocon Ointment, 0.1%	Mometasone furoate
Florone Ointment, 0.05%	Diflorasone diacetate
Halog Ointment/Cream, 0.1%	Halcinonide
Lidex Cream/Gel/Ointment, 0.05%	Fluocinonide
Maxiflor Ointment, 0.05%	Diflorasone diacetate

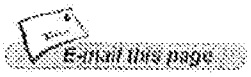
Maxivate Ointment, 0.05%	Betamethasone dipropionate
Psorcon Cream 0.05%	Diflorasone diacetate
Topicort Cream/Ointment, 0.25%	Desoximetasone
Topicort Gel, 0.05%	Desoximetasone
CLASS 3 - Upper Mid-Strength	
Aristocort A Ointment, 0.1%	Triamcinolone acetonide
Cutivate Ointment, 0.005%	Fluticasone propionate
Cyclocort Cream/Lotion, 0.1%	Amcinonide
Diprosone Cream, 0.05%	Betamethasone dipropionate
Florone Cream, 0.05%	Diflorasone diacetate
Lidex-E Cream, 0.05%	Fluocinonide
Luxiq Foam, 0.12%	Betamethasone valerate
Maxiflor Cream, 0.05%	Diflorasone diacetate
Maxivate Cream/Lotion, 0.05%	Betamethasone dipropionate
Topicort Cream, 0.05%	Desoximetasone
Valisone Ointment, 0.1%	Betamethasone valerate
CLASS 4 - Mid-Strength	
Aristocort Cream, 0.1%	Triamcinolone acetonide
Cordran Ointment, 0.05%	Flurandrenolide
Derma-Smoothe/FS Oil, 0.01%	Fluocinolone acetonide
Elocon Cream, 0.1%	Mometasone furoate
Kenalog Cream/Ointment/Spray, 0.1%	Triamcinolone acetonide
Synalar Ointment, 0.025%	Fluocinolone acetonide
Uticort Gel, 0.025%	Betamethasone benzoate
Westcort Ointment, 0.2%	Hydrocortisone valerate
CLASS 5 - Lower Mid-Strength	
Cordran Cream/Lotion/Tape, 0.05%	Flurandrenolide
Cutivate Cream, 0.05%	Fluticasone propionate
DermAtop Cream, 0.1%	Prednicarbate
DesOwen Ointment, 0.05%	Desonide
Diprosone Lotion, 0.05%	Betamethasone dipropionate
Kenalog Lotion, 0.1%	Triamcinolone acetonide
Locoid Cream, 0.1%	Hydrocortisone butyrate
Pandel Cream 0.1%	Hydrocortisone probutate
Synalar Cream, 0.025%	Fluocinolone acetonide
Uticort Cream/Lotion, 0.025%	Betamethasone benzoate

Valisone Cream/Ointment, 0.1%	Betamethasone valerate
Westcort Cream, 0.2%	Hydrocortisone valerate
CLASS 6 - Mild	
Aclovate Cream/Ointment, 0.05%	Alclometasone dipropionate
DesOwen Cream, 0.05%	Desonide
Synalar Cream/Solution, 0.01%	Fluocinolone acetonide
Tridesilon Cream, 0.05%	Desonide
Valisone Lotion, 0.1%	Betamethasone valerate
CLASS 7 - Least Potent	
Topicals with hydrocortisone, dexamethasone, methylprednisolone and prednisolone	

Updated July 2004

Related links

- Topical steroids
- Internal use of steroids
- Methods of using topical steroids
- Side effects of topical steroids
- Tips for using topical steroids



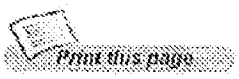
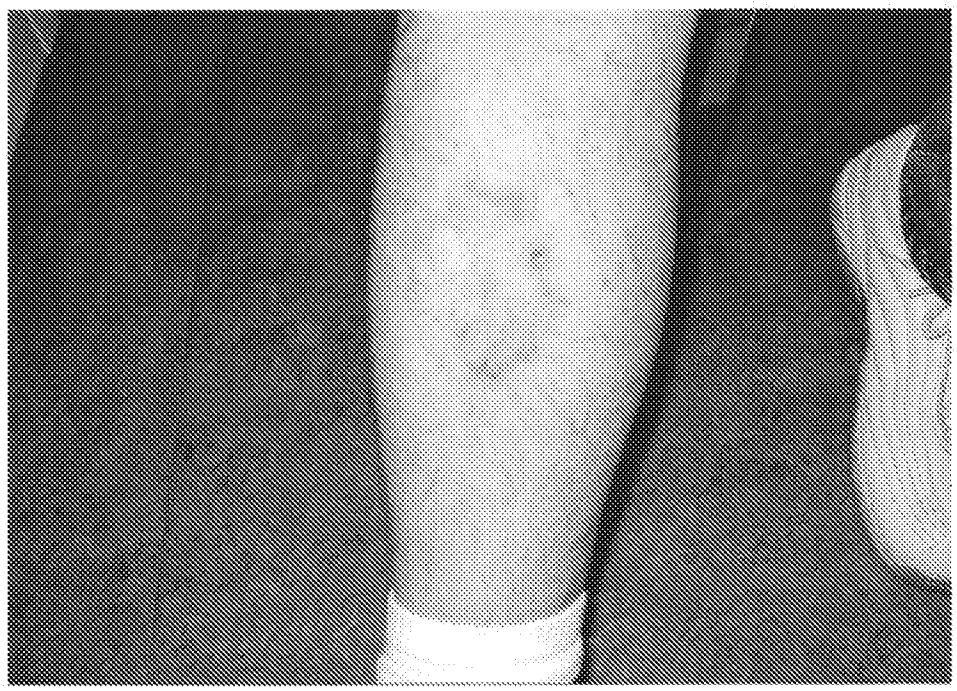
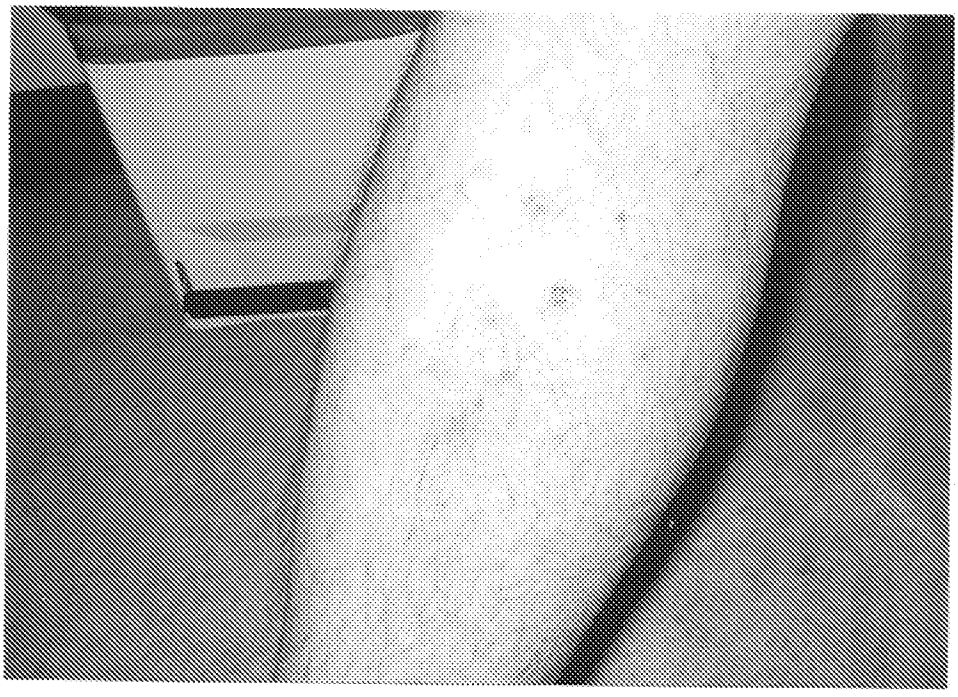


EXHIBIT D



Before Treatment



After Treatment

PATIENT INFORMATION [redacted]

PATIENT ________ DATE OF BIRTH ________ DATE ________ AGE 62 SEX M

NAME ________ SPOUSE'S NAME ________

RESPONSIBLE PARTY IF PATIENT IS A [redacted]

ADDRESS [redacted] NUMBER STREET CITY OR TOWN ZIP CODE PHONE [redacted]

EMPLOYER ________ POSITION ________

EMPLOYER'S ADDRESS ________ PHONE [redacted]

FAMILY PHYSICIAN ________

REFERRED BY ________

INSURANCE INFORMATION ________ SOCIAL SECURITY NUMBER ________

SUBSCRIBER ________ RELATIONSHIP ________

BLUE SHIELD BLUE SHIELD MASTER HEALTH PLUS ☐ MEDICARE ☐ MEDEX ☐

TUFTS ☐ PILGRIM HEALTH ☐ OTHER ☐ INS. CO NAME ________

PRIMARY INS. CERT. # ________ SECONDARY INS. CERT. # ________

[illegible handwriting] back, arm, legs, LGH. Nm NSC.

11-04

Here for evaluation of multiple lesions back, arms, legs.

PE - Complete detailed skin exam neg. except for the following neoplastic lesions.

1. large fiber epithelial papilloma, (R) arm anterior.
2. FEP, (R) hip.
3. Probable small SK vs. AK, inferior to (L) knee crease.
4. Multiple axillary bilateral SKs. Note: there is also 10 toenail onychomycosis

Note: there is also scattered benign SKs of back.

Plan - 1. RTC 2 wks. for simple excision of (R) arm, (R) hip FEP's, and D&C of (L) lower leg posterior, ? AK - Note: this is just inferior to knee crease.

11-25

Here for excision, pt. made aware of possibility of keloidal scarring and/or pigmentary change

Plan - 1. Simple excision of 1.1cm (R) arm FEP, 0.6cm (R) hip FEP, and D&C of (L) lower leg AK vs. SK, 2% plain xylo, STE.

2. RTC 6 wks. for treatment of multiple neck skin tags under emla.

12-09

Phone conf. advised of path DX.

1-06

15+ axillary skin tags treated with light electrocautery. RTC PRN.

8-12

Pt. may have had "tick bite" 4 or 5 days ago. Was seen at LMH ER and pt. states was given appropriate anti Lyme medication. Patch of dermatitis which is concerning to pt. remains, (R) lower leg although may have closed up somewhat.

PE - Complete detailed skin exam neg. except for small scaly plaque, (R) lower leg, and pigmented lesion, (L) mid back.

IMP - 1. ? Lyme Disease although was adequately treated at LMH ER.

2. Patch of dermatitis, (R) lower leg.
3. Pigmented (L) mid back lesion.

Plan - 1. Start ApexiCon E Cream BID. Call if not clear 2 to 3 wks.

(cont'd)

8-12
Plan - 2. D&C or (L) mid back pigmented lesion, 2% plain xylo, Dermpath.
3. Call path 1 wk.

8-18
Phone conf. advised of path DX. Pt. states leg eruption still present although improved. Advised to continue treatment and if not clear 1 to 2 wks. to call.

9-26
Complaining of new eruption essentially asymptomatic, (L) lower leg X 3 wks. Also irritated lesion, (L) side of neck.
PE - Small FEP (L) side of neck. ? vasculitis, (L) lower leg.
Plan - 1. Simple excision of 0.3cm (L) side of neck ? FEP, 2% plain xylo, Dermpath.
2. 3mm punch BX from (L) lower leg ? vasculitis, 2% plain xylo, Dermpath.
3. RTC 7 days to discuss lab results and begin treatment.

10-3
Pt. advised of path DX. No change since last visit.
PE - Annular plaque remains without scale, (R) lower leg.
Plan - 1. Change to Bactroban Cream BID samples.
2. Start ApexiCon Ointment BID.
3. Lyme titer.
4. RTC 8 days.

10-6
Phone conference. Advised of neg. lyme titer.

10-7
Message left that pt. is doing well. Will be seen as appointed on Tuesday.

10-11
Very pleased with progress.
PE - Marked to complete clearing of eruption on lower leg.
Plan - 1. Taper then DC ApexiCon.
2. Call and return if not completely clear 4 wks.

10-24
Eruption was somewhat clear but has recurred of meds.
PE - Bright red erythematous eruption on (R) lower leg. (L) lower leg clear as is remainder of skin.
Plan - 1. Refer for second opinion.
2. Temporarily continue to stay off ApeixCon.

10-28
Pt. advised of path DX, namely tinea.
PE - Bright red erythematous eruption remains, still visible on (R) lower leg.
Plan - 1. Trial of Desonide and Lotrimin/Ertaczo samples BID.
2. RTC 2 wks. - call progress 5 days.

11-2
Phone conference. Pt. reports marked improvement.

11-10
F/U inflammatory tinea of (R) lower leg. Pt. very pleased with progress.
PE - Marked to complete clearing of eruption, (R) lower leg.
Plan - 1. Has been using Desonide/Lotrimin combo - may taper to once daily for 1 wk. and then
2. Call immediately if recurs. Otherwise RTC PRN.